Exhibit A

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17

Mild Head Injury in Sports: Neuropsychological Sequelae and Recovery of Function

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Almost 45 years ago, Holbourn (1943) postulated the existence of "shear strain" of axonal and dendritic tissue, secondary to what is now described as rotational, acceleration/deceleration head trauma (Gennarelli, 1986; Gennarelli et al., 1981; Ommaya and Gennarelli, 1974). Only in the past seven years, however, has this theory of neurological impairment been utilized to explain the symptoms of mild head injury (postconcussive syndromes) (Alves et al., 1986; Boll and Barth, 1983). The symptoms of mild head injury usually include headaches, dizziness, memory loss, nausea, diplopia, tinnitus, personality change (irritability), attentional deficits, and information processing impairment. Although factors affecting rate and extent of recovery and persistence of symptoms continue to generate scientific debate, the popular press has characterized mild head injury as a "silent epidemic," which has subsequently drawn more attention to the potentially devastating cognitive, emotional, and economic consequences of this trauma (Barth et al., 1983; Rimel et al., 1981). In general, the public and scientific community have been slow to react to this rather common pathology, which results in hospitalization of close to 300,000 patients per year in the United States alone. Moreover, many more mild injuries occur that do not require extensive medical observation and intervention (Kraus et al., 1984).

Similar to the general public, competitive athletes are at risk for head injuries, but the evolving field of sports medicine is usually ahead of the general public in recognizing potential health problems for athletes and implementing corrective or protective strategies. For example, professional race car drivers

were the first to wear seatbelts, and professional motorcycle racers wore helmets long before the general public took advantage of these protective devices.

In most sports, great efforts are made to evaluate and eliminate the potential for what is viewed as *significant or severe* head trauma. This has not, however, been the case with most sports-related *mild* head injuries. Until recently very little has been written about mild head trauma in sports because it was not seen as a major problem. Athletes typically do not complain about "minor injuries" because doing so would be a sign of weakness, and worse, might result in elimination from participation in upcoming competition. The ramifications of admitting to any injury include the possibility of losing one's position and scholarship (in collegiate sports); letting down the team, coaches, school, and fans; missing an opportunity to display one's skills to the professional agents and scouts; and being seen as a failure by oneself and one's friends and family. Because of our somewhat controversial and still limited knowledge of the effects of mild head injury and the negative consequences of athletes' admission of such problems, it is not surprising that mild head injury has not been a principal concern of most athletes or their institutions and organizations.

HEAD TRAUMA IN BOXING

Although the potential effects of head injuries have generated little attention in some sports, moderate and severe head trauma have been studied in some depth in sports such as boxing (Morrison, 1986). Most studies of sports-related head injury have focused on boxing because it stands alone among other contact sports in having as its goal rendering opponents unconscious and helpless through successive blows to the head (Council on Scientific Affairs [AMA], 1983; McCunney and Russo, 1984). The earliest description in the literature on neurological, cognitive, and behavioral impairment resulting from boxing was provided by Martland (1928). In an article entitled "Punch Drunk," he delineated various neurological symptoms that characterize the syndrome, including mild confusion and an unsteady gait early on, which progresses to increased speech and motor latencies as well as upper-extremity and head tremors. Martland has stated that eventually this syndrome is characterized by a movement disorder similar to that seen in Parkinsonian patients, usually involving an extremely unsteady gait and considerable mental decline. Diffuse cerebral atrophy often occurs and has been variously labeled as "chronic boxers' encephalopathy" (Serel and Jaros, 1962), "dementia pugilistica" (Lampert and Hardman, 1984), and "traumatic boxers' encephalopathy" (Mawdsley and Ferguson, 1963).

Mechanisms and Neuropathological Consequences of Acute Brain Damage

Lamper and Hardman (1984) describe four basic mechanisms that account for the neuropathological changes evidenced in acute brain damage from boxing and other contact sports. These include (1) rotational acceleration; (2) linear acceleration; (3) carotid injuries; and (4) deceleration upon impact. The

authors state that rotational or angular acceleration, poses the most danger to a fighter, and typically occurs when he is groggy and most vulnerable. This pathological mechanism involves sudden acceleration of the head with subsequent rotational movement of the brain inside the skull, leading to the stretching and snapping of blood vessels. Paradoxically, protective head gear, such as the type worn by amateurs, tends to increase the inertial force of angular acceleration by increasing the surface size around the head (Timperley, 1982). Complications and lesions such as diffuse axonal injuries, subdural hematomas, and intracerebral hemorrhages may result from rotational force, depending upon the characteristics of the impact.

The second mechanism described is that of linear or translational acceleration, which results in focal cerebellar ischemic lesions (Lampert and Hardman, 1984). These lesions "develop a few days after repetitive trauma and their severity increases proportional to the number of subconcussive blows" (p. 2678).

Carotid artery injury resulting from blows to the neck can cause reflex hypotension and a subsequent brief period of dizziness due to diminished blood flow to the brain. This is a dangerous state for the boxer, who then is more vulnerable to significant head acceleration from an opponent's blow.

The final mechanism described by Lampert and Hardman is that of impact deceleration, which can occur when the dazed or knocked-out boxer hits the canvas or rope posts. This typically can result in occipital coup and frontotemporal contrecoup lesions. Clinically though, whatever the injury mechanism may be, these types of neurotrauma are complicated by the body's natural responses such as edema, ischemia, and possibly herniation of brain tissue within the skull (Lampert and Hardman, 1984; Serel and Jaros, 1962).

Severity and Long-Term Effects

An important factor contributing to the severity of head trauma in boxing, particularly as this relates to long-term residual deficits, is the cumulative effects of multiple blows to the head, not necessarily resulting in knockouts. Casson et al. (1982) studied 10 professional boxers shortly following a knockout with electroencephalography (EEG), computed tomography (CT), and neurological examinations. These professional athletes, 20–31 years of age, had engaged in anywhere from two to 52 bouts. According to these authors, "All the boxers had mild head injuries by clinical criteria, with duration of loss of consciousness and posttraumatic amnesia of less than two minutes" (p. 172). Testing results, however, indicated that one boxer had an abnormal neurological examination, another two had abnormal EEGs, and five evidenced clearly abnormal CT scans with mild to moderate cerebral atrophy. These authors suggested that abnormalities that they detected were not due to the number of knockouts because no boxer had even been knocked out more than twice. Instead, they indicated that multiple subconcussive blows to the head was a much more plausible etiology.

Kaste et al. (1982) in one of the few neuropsychological studies, examined eight amateur and six professional boxers. Results showed that 86% of these

boxers performed in the mildly impaired range on the Trail Making Test from the Halstead–Reitan Neuropsychological Test Battery, and two professional boxers demonstrated more severe neuropsychological difficulties. One boxer obtained very poor scores on the Digit Symbol Test from the Wechsler Adult Intelligence Scale, the Benton Visual Retention Test, and the Trail Making Test, whereas the other had performed poorly on the Wisconsin Card Sorting Test and the association learning subtest of the Wechsler Memory Scale. CT scans revealed "brain atrophy in 3 of 6 professionals and 1 of the 8 amateurs" (p. 1187). The authors concluded that the effects of repeated concussions are cumulative, and beyond a yet to be determined number of concussions, the neuropathology is likely to be irreversible.

Casson et al. (1984) studied 18 active and former boxers who underwent neurological examination, neuropsychological testing, EEG, and CT. They excluded subjects with a history of neurological impairment, psychiatric difficulties, or drug and alcohol abuse. All fighters, in addition, were active in the post-World War II era, indicating that they fought during a time when increased medical supervision became an issue of public concern. However, subjects in this study were not selected at random, limiting the generalizability of the results. According to these authors, 87% of all boxers exhibited abnormal findings on at least two of the four examinations. Each boxer scored within the impaired range on more than one neuropsychological measure which included the Trail Making Test, Digit Symbol Test, Wechsler Memory Scale, and recall of the Bender Visual–Motor Gestalt figures. They found that the percentage of abnormal neuropsychological test scores correlated significantly with abnormal CT scans, age, and the number of professional fights. These authors concluded that their study lends "further support to the direct relationship between length of professional boxing career and the presence of brain damage" (p. 2666).

Mild Head Injury in Boxing

Relatively little is known about *mild* head injury in boxers although some previously cited studies such as that by Casson et al. (1982) refer to such trauma. Amateur boxers have generally had fewer bouts, rounds, and knockouts, and they usually weigh less and are more closely supervised than their professional counterparts. Because these factors have been shown to be directly related to severity (as well as chronicity) of neurobehavioral trauma, amateur and young professional boxers are the main focus of such mild head injury studies.

An early investigation by Blonstein and Clarke (1957) examined athletes who were knocked out more than once, or suffered severe concussions in the London Amateur Boxing Association during a seven-month period. They found that only 58% ($N = 29$) of all boxers active at that time fit their criteria. EEG and neurological examinations did not detect abnormalities, although the theory "that deleterious cerebral changes may occur in amateurs after an interval is a possibility that has by no means been excluded by this study" (p. 363). They raised an additional point, however, which is of some interest: that is, the importance of assessing retrograde and anterograde amnesia in boxers who

were not rendered unconscious. These authors suggested that a classification of boxing head injuries according to degree of amnesia was potentially useful. Duration and severity of traumatic amnesia proved helpful in understanding and predicting the sequelae of head injury, and might be particularly useful with regard to mild head injury in boxing.

A more recent study examined neuropsychological, neurological, and EEG results in 53 former amateur boxers and a control group of 53 former amateur soccer players (Thomassen et al., 1979). Significant differences were not found between soccer players and boxers in the EEG or neurological examinations performed. Neuropsychological data showed that boxers evidenced significantly more dysfunction than the control group on measures of left-handed motor function, visuospatial intelligence, expressive speech, logical memory, phonetic analysis of numbers, and synthesis of letters in simple and complex words. Yet, after results were statistically corrected for age, education, and vocabulary, the only significant differences remaining between boxers and controls concerned left-handed motor dysfunction.

CT scan, EEG, and neurological evaluations of professional boxers in Italy were completed to facilitate early detection of cerebral atrophy. This study found significant correlations between CT scan and EEG findings but no evidence that neurological assessment was of diagnostic benefit (Sironi et al., 1982). Young boxers' career successes and failures were directly related to CT scan and EEG results (i.e., the higher the number of knockouts, the more impairment on these tests). Years of boxing activity and number of matches did not predict cerebral dysfunction.

Three very recent studies of young amateur and professional boxers utilized standardized neuropsychological test procedures in addition to other medical procedures to determine the effects of what may be considered mild to moderate head trauma (Brooks, 1987; Levin et al., 1987a; McLatchie et al., 1987). In the McLatchie et al. study of 20 active boxers, 1 had an abnormal CT scan, 7 had impaired neurological exams, 8 demonstrated abnormal EEG, and 9 out of 15 had deficient neuropsychological examinations. Because the boxers performed significantly more poorly than controls on several neuropsychological measures, the authors concluded that neuropsychological assessment is the most sensitive measure of neurologic dysfunction.

Brooks et al. (1987) and Levin et al. (1987a) on the other hand, found no significant neuropsychological deficits in their samples of amateur and young professional boxers and matched controls. They suggest that their results should be intrepreted cautiously because absence of neuropsychological deficits could be due to such factors as low frequency and duration of ring exposure and knockouts as well as the possibility of the delayed appearance of degenerative disorders such as Alzheimer's disease.

It is unclear what effect mild head injury has on immediate and long-term neuropsychological functioning in amateur boxers. Longitudinal studies of a wide range of amateur boxers with varying skills, levels of success, frequency of ring exposure, head and neck contacts, and knockouts must be initiated to address this question. Johns Hopkins University, supported by the United States Olympic Committee, is presently carrying out such research and hopes

to control for additional factors such as premorbid cognitive and psychosocial functioning.

FOOTBALL

Empirical attention has also been paid to football-related head trauma; however, like boxing, few controlled prospective studies are available in the scientific literature. Epidemiological, descriptive, retrospective, and case studies provide the basis for most of our understanding of football head injury.

The mechanisms of these injuries are similar to boxing trauma described by Lampert and Hardman (1984), and severity of impairment often appears to be related directly to number and recency of previous blows to the head or acceleration/deceleration injuries. Case studies such as those reported by Harbaugh and Saunders (1984) and Schneider (1973) demonstrate devastating cerebral injury characterized by widespread edema, midline shift, herniation, hemorrhage, and anoxic changes secondary to what appears to be relatively mild trauma if such trauma follows closely on the heels of previous mild head injury.

A retrospective study by Gerberich et al. (1983) of 103 Minnesota high school football teams, comprised of 3,063 varsity players who responded to mailed questionnaires, revealed a 19% loss of consciousness or awareness rate during the 1977 season. Sixty-nine percent of these concussed players returned to athletic competition the same day, but postconcussive symptoms were reported by some players up to nine months post season. Permanent disability was noted in six cases and "players with a prior history of loss of consciousness had a risk of loss of consciousness four times that of the player without a prior history" (p. 1370).

In 1979, Torg, Truex, Quedenfeld, Burstein, Spealman, and Nichols published results from the National Football Head and Neck Injury Registry which began collecting data in 1971 on football injuries that involved "hospitalization for more than 72 hours, surgical intervention, fracture-dislocation, permanent paralysis or death" (p. 1477). There were 1,129 injuries that met these severe head and neck injury criteria, and over that time period the rate of intracranial hemorrhage decreased, presumably owing to the development of better head and face protective helmets. However, they also reported a concomitant increase in the number of cervical spine injuries due to a recently outlawed tackling technique known as *spearing,* involving use of the head and helmet to knock down a player.

A prospective study of head and neck injuries in 342 football players over an eight-year period at the University of Iowa (Albright et al., 1985) established an incidence of 175 injuries in 100 players during the length of the study.

> [Players with previous] histories of head or neck injuries or abnormalities of the cervical spine (on prescreening) were twice as likely to have head or neck injuries at some point in their college careers as those players with a normal screening examination, [and] . . . the probability of subsequent head or neck injury escalated sharply following a single incident. (p. 147)

According to Maroon et al. (1980), in their review of 47 years of National Collegiate Athletic Association and American Football Coaches Association data collection efforts, most severe football head injuries occur during defensive blocking or tackling maneuvers. They also point out, as do Harbaugh and Saunders (1984), that most team physicians apply what is referred to by Schneider (1973) as *Quigley's rule,* when evaluating potential dangers from cerebral insult. This rule proposes that athletes should discontinue active participation in sports after receiving three cerebral concussions. This rule is supported by data gathered by Gronwall and Wrightson (1975) as well as others, who contend that cognitive deficits are cumulative in successive concussions. Maroon et al. (1980) go on to underscore that neurological impairment that is documented by CT or other radiological measures "may be enough to strongly discourage or forbid further football participation" (p. 427).

Mild Head Injury in Football

Mild head injury in football has only recently been considered a problem requiring scientific investigation. Most football players report having one or more mild concussions or "dings" over their careers. These injuries are often characterized by a change in (but not loss of) consciousness, as well as confusion, retrograde or posttraumatic amnesia, or immediate memory loss, yet these athletes usually continue to play (Meggysey, 1970; Yarnell and Lynch, 1973). These symptoms are often complicated by headache and/or dizziness as well as other postconcussion symptoms (Cook, 1969).

Several attempts have been made to classify cerebral concussion in athletes; these systems have been summarized by Nelson et al. (1984). Torg (1982) has described four grades of mild and moderate head trauma which range from symptoms of having one's "bell rung" (i.e., short-term confusion, unsteady gait, being dazed, and mild posttraumatic amnesia), to immediate and transient loss of consciousness with the above symptoms. Nelson et al. (1984, p. 104) have argued for a concussion classification system for athletes that would focus on five grades of mild head injury, from grade 0, indicating no loss of consciousness but confusion and subsequent difficulty concentrating; to grade 4, characterized by loss of consciousness for more than one minute (yet no coma), headache, cloudy sensations, possible irritability, confusion, and dizziness during recovery.

Although there were no prospective neuropsychological studies of mild head trauma in football players until our own (described later), the recent surge of concern regarding mild head injury in the clinical population has generated interest in high school and college communities, with particular focus on early identification, recovery, return to practice and athletic competition, and improvement of equipment, rules, and coaching techniques. More recently, it has been proposed not only that team physicians implement Quigley's rule, but also that resumption of athletic activities after one mild head injury be predicated on a total resolution of postconcussive symptoms (Hugenholtz and Richard, 1982). In addition, Yarnell and Lynch (1973), in their article entitled "The 'ding': Amnestic States in Football Trauma," based on observations and case

studies, concluded that evaluation of memory is a key aspect in the assessment of mild concussion. They refine this concept by suggesting that

> one should not ask [concussed players] questions requiring immediate memory or cognition (digit recall, simple arithmetic, reverse spelling, etc.). Rather, items concerning recently experienced or consolidated events are [more appropriate assessment strategies] ("Where did you go after you left the playing field? What are some of your plays and assignments for this game?"). (Yarnell and Lynch, 1973, p. 197)

HEAD TRAUMA IN OTHER SPORTS ACTIVITIES

Although head trauma tends to occur more frequently as a result of participation in such sports as boxing and football, athletes who engage in a variety of other competitive physical activities are also at risk for sustaining craniocerebral injuries. Head trauma appears to be the most common, as well as the most severe, type of injury in the equestrian sports (Bixby-Hammett, 1983). Riders who do not wear helmets are at additional high risk for sustaining significant head trauma. Foster et al. (1976) describe five cases of brain damage in British National Hunt jockeys, one of whom died a number of days after being admitted to a neurological center in a coma. All of the jockeys had histories of previous concussive injuries sustained during race-riding. These authors conclude that "National Hunt jockeys are exposed to frequent and often severe unrecorded concussive head injury, and that this can result in brain damage and temporal-lobe epilepsy and the other features recognized as post-traumatic encephalopathy" (Foster et al., 1976, p. 983). As is the case for authors reporting on boxing in the United States, Foster and his colleagues call for stricter safety regulations in order to prevent individuals from sustaining repeated, and rather severe head trauma. Ilgren et al. (1984) reviewed a number of cases in which horse riding accidents resulted in neurotrauma. These authors have observed that this cumulative trauma effect occurs in equestrians as well and results in progressive encephalopathy.

Harris (1983) studied 126 winter sports injuries, occurring over a four-year period, at a neurosurgical medical clinic in an American winter resort area. The sample included 82 individuals who sustained concussions while skiing. The most severe neurotrauma appeared to result from one of four mechanisms of injury: (1) colliding with trees; (2) colliding with other skiers; (3) colliding with large boulders or ski-lift equipment; and (4) performing aerial maneuvers. Harris states that the most dangerous collisions occurred at high speeds, with the skier being abruptly decelerated upon impact. Consequently, the extent and nature of the neuropathology sustained typically fell more toward the severe end of the injury spectrum.

The literature on head trauma resulting from participation in other sports has typically described cases of significant and extensive neuropathology often leading to death (with little mention of mild head injury). These athletic activities include squash (Clement and Fairhurst, 1980), rugby (Roy, 1974), lacrosse (Rimel et al., 1983), and ice hockey (Fekete, 1968; Kraus et al., 1970). Many

of these authors conclude that special safety helmets should be developed, and worn by players and that serial concussions should be avoided.

MAGNITUDE OF THE SPORTS-RELATED HEAD TRAUMA PROBLEM

Although head trauma in sports is a serious problem (see further reviews by Kraus and Conray, 1984), it pales in comparison to head trauma unrelated to sports in the general clinical population. In fact, in one epidemiological study, only 2.7% of 1,900 significant head injuries over a five-year period in Glasgow, Scotland, were sports related (Lindsay et al., 1980). Within sports themselves, significant head injuries seem to occur quite infrequently, and only a small percentage of players suffer such trauma (Bruce et al., 1984). For example, only 1% of 19,413 high school football players in southern California sustained significant head injuries in 1961 (Alley, 1964), and the incidence of such injury appears to have decreased over time.

The incidence and outcome of sports-related mild head trauma have not been sufficiently investigated, and empirical examination of head injury in sports remains important in order to (1) continue reducing the rate and severity of head injury in this arena; (2) ensure rapid and complete recovery; (3) determine the seriousness of the mild head trauma and repeated injury in sports; and (4) utilize sports injury as a "laboratory" model for acceleration/deceleration mild head injury in the general population.

THE UNIVERSITY OF VIRGINIA PROSPECTIVE STUDY OF MILD HEAD INJURY IN FOOTBALL

For the above reasons, in June 1982 we began a four-year prospective study of football-induced mild head injuries, which was sponsored by a grant from the Pew Memorial Trusts. The objectives of the study were to (1) estimate the incidence of football-induced head injuries and determine the extent and nature of neuropsychological and psychosocial deficits of injured players; (2) establish a recovery curve for players with mild head injury and develop guidelines as to when players can resume normal activities, including football; (3) identify the personal and football-related factors predisposing players to the risk of head injury; and (4) evaluate the longer-term neuropsychological and psychosocial consequences of sustaining more than one head injury during players' college careers.

Data were collected on a total of 2,350 players at 10 universities including the Ivy League (Brown, Columbia, Cornell, Dartmouth, Harvard, Pennsylvania, Princeton, and Yale), the University of Pittsburgh, and the University of Virginia. Players who sustained a mild head injury during this study were evaluated for cognitive and psychosocial dysfunction through the use of neuropsychological techniques and self-report questionnaires up to four times after injury. Mild head trauma was defined as a change in or loss of consciousness for less than two minutes and immediately demonstrating attentional/memory

Table 17–1 Classification of Players Studied

Total number of players	2350
Postinjury protocol:	
Head injuries	195
Orthopedic injuries	59
Student controls	48

problems, as suggested by Yarnell and Lynch (1973). Team physicians and trainers documented 195 injuries involving 182 players during the study. Twelve players suffered two injuries, and one player sustained three injuries.

In addition to head-injured players, 59 players sustaining mild orthopedic injuries and 48 male college students were tested using the same protocol. These two control groups were employed to provide an estimate of the learning effects of repeated testing (normal testing behavior) and to assess the possible cognitive and psychosocial consequences of trauma per se as compared to head trauma.

The tables that follow present some of our initial data analyses. Tables 17–1 and 17–2 show the number of players tested and their characteristics, based on the entire population of football players studied. Of special note is section C of Table 17–2 which indicates that approximately 42% reported a history of at least one minor head injury prior to participation in the study. Over 11% reported two prior minor head injuries, and 11.6% had sustained three or more such injuries. The consequences of the history of previous head injury are currently being explored in our continuing analysis of the data.

Table 17–2 Player Characteristics (N = 2350)

	Mode	Percent
A. *Age and year in school*		
Age	19	48.4[a]
Year in school	Sophomore	56.7[a]
B. *Position Played*		
Offensive line		23.01
Receivers		10.18
Running back		11.39
Quarterback		5.72
Defensive line		16.93
Linebackers		11.89
Defensive back		16.93
Other (special teams)		3.95
C. *History of previous head injury*		
None		57.54
One		19.52
Two		11.34
Three or more		11.60

[a]Percent of total series who are at model age and year in school.

Table 17–3 Time When Injury Occurred (N = 195)

	Percent
Game	
First quarter	15.4
Second quarter	12.7
Third quarter	14.3
Fourth quarter	13.2
Total	55.6
Practice	
First session	33.2
Second session	9.8
Third session[a]	1.4
Total	44.4

[a]Only the University of Pittsburgh engaged in three daily practice sessions during the preseason practice period.

Table 17–3 indicates that there is no apparent game quarter in which players are at greater risk for mild head injury. Nearly 56% of the injuries occurred in the game situation, suggesting that players are at slightly more risk for injury during a game situation as compared to practice sessions. The vast majority of practice-related injuries occurred during the first practice session of the day. Our initial finding is that most practice injuries do occur during the preseason session and may be releated to conditioning and/or coaching factors, whereas during the season most injuries occur in game situations.

Table 17–4 shows the injured players' positions. The only group of players that seem to be at somewhat greater risk appear to be those who perform special team functions (e.g., kickoff and punt return teams). The injury rates for other player positions seem consistent with the distribution of all players across the various team positions (see Table 17–2). We should point out, however, that it is possible that there is a differential risk to players in certain positions, depending on the number of plays in a game situation in which those specific positions may be involved. As expected, tackling and blocking are the primary activities of players when they are injured (see Table 17–5). There is

Table 17–4 Injured Player's Position (N = 195)

Position	Percent
Offensive line	23.2
Receiver	7.9
Running back	12.9
Quarterback	2.3
Defensive line	13.6
Linebacker	10.7
Defensive back	15.8
Other	13.6

Table 17–5 Injured Player's Activity (N = 195)

Activity	Percent
Running and tackled	14.8
Tackling opponent	18.1
Being blocked	17.0
Blocking opponent	25.8
Other (includes practice drills)	24.3

slightly more risk for the player who is blocking or tackling an opponent (nearly 44%), as compared to the player who is being blocked or tackled (nearly 32%). The majority of "other" activities (comprising 24% of injured players' activities) are largely special team functions as discussed above, and injuries occurring during practice drills.

Table 17–6 lists the primary mechanism of injury classified by impact to the head versus no apparent impact to the head. Over 54% of all injuries involved direct impact to the head, whereas 34.6% involved no impact to the cranium. In 10.8% of the cases, the mechanism of injury was unclear (i.e., there was no identifiable collision that could be identified). In injuries involving direct impact to the head, the majority were helmet-to-helmet impacts (21.1%). Almost 12% of trauma involved a collision to the head in which an opponent's torso or body struck the head, 9.19% of the players were kicked in the head, and 2.16% were kneed in the head. The primary nonimpact category was collision with another player, which did not involve direct impact to the head but which probably involved some form of rotational injury. We are currently reviewing the narrative descriptions of the injuries to see if we can provide more detail interpreting these broad categories of mechanism of injury.

Tables 17–7 to 17–9 present initial findings from our analyses of neuropsychological test scores which were gathered at preseason, 24 hours post injury, 5 and 10 days post trauma, and at post season. Assessment procedures included the Trail Making Test A and B from the Halstead–Reitan Neuropsychological Test Battery (Reitan and Davison, 1974), Aaron Smith's Symbol Digit Test (Smith, 1973), and the Paced Auditory Serial Addition Task (PASAT) (Gronwall and Sampson, 1974; Gronwall and Wrightson, 1980). The

Table 17–6 Mechanism of Injury (N = 195)

Impact to head	
Helmet-to-helmet	21.08%
Collision to head	11.89
Kick to head	9.19
Head struck ground	3.78
Head first tackle	3.24
Knee to head	2.16
Head-to-torso	2.16
Forearm to chin	1.08
Total	54.6
No impact to head	
Collision with other player	24.40%
Hard tackle	4.86
Hard block	1.62
Speared on ground	1.62
Speared in air	1.08
Total	34.6
Unclear (no identifiable impact)	
Total	10.8%

Table 17–7 Changes in PASAT 4 Scores of Head-Injured Players and Student Controls (Mean Score and Percent Change)

	Head injured		Student controls	
Time of study	Mean score	Percent change[a]	Mean score	Percent change[a]
Preseason	66.3%		66.8%	
		5.3%		17.7%
24 hours	69.8		78.7	
		14.8		7.6
5 days	80.1		84.6	
		6.5		4.1
10 days	85.3		88.1	
		0.7		1.1
Postseason	84.7		89.1	

[a]Percent change reflects the percent increase in PASAT 4 scores at the time of the study, compared to the score at the preceding time of study (e.g., 24 hours post injury compared to preseason; 5 days post injury compared to 24 hours, etc.).

Trail Making Test is a measure of sustained attention and concentration, which requires sequential problem solving and ability to keep two things in mind simultaneously. The Symbol Digit Test is an alternative to the Wechsler Adult Intelligence Scale Digit Symbol subtest and requires psychomotor problem solving and visual perceptual abilities. Gronwall's PASAT is a task involving rapid presentation of auditory numeric material for complex mental manipulation and requires a high level of attention, concentration, and immediate recall memory.

Table 17–7 shows the mean percentage of correct responses at each time of study and changes in mean scores of head-injured players and student controls on subtest 4 of the PASAT (1.2-second interval between numbers). These changes (reported as percent change) indicate increases in PASAT 4 at the time of study compared to the score at the preceding study time (e.g., 24 hours v. preseason, 5 days vs. 24 hours, etc.). Tables 17–8 and 17–9 present the ratios of average test score differences and their standard errors (average test score difference divided by standard deviation) for head-injured players (Table 17–8) and student controls (Table 17–9). The ratios shown in Tables 17–8 and 17–9 can be interpreted as statistically significant if their absolute value is greater

Table 17–8 Ratios of Average Test Score Differences and Standard Error of Differences for Head-Injured Players

Difference between:[a]	Trail A	Trail B	Symbol digit	Pasat 3	Pasat 4
Pre-Sn—24 hours	−2.39	−2.46	.54	1.13	1.93
24 hours—5 days	−5.04	−5.29	6.39	4.44	5.73
5 days—10 days	−1.84	−3.00	3.53	2.83	3.33
10 days—Post-Sn	−0.02	1.99	−2.59	0.12	0.42

[a]*Later* score minus *earlier* score.

Note: Ratios refer to average test score difference as compared with previous examination (see column on left) divided by standard deviation.

Table 17–9 Ratios of Average Test Score Differences and Standard Error of Differences for Student Controls

Difference between:[a]	Trail A	Trail B	Symbol digit	PASAT 3	PASAT 4
Pre-Sn—24 hours	−3.79	−2.55	2.46	3.04	4.04
24 hours—5 days	−1.47	−2.59	2.97	1.73	2.19
5 days—10 days	−2.05	−2.54	1.06	0.99	1.39
10 days—Post-Sn	−0.05	0.38	−0.15	2.45	0.42

[a]*Later* score minus *earlier* score.
Note: Ratios refer to average test score differences divided by their standard deviation.

than or equal to 1.96. The changes in PASAT subtest 4 scores shown in Table 17–7 display a rather typical pattern when comparing head-injured players and student controls. The change from preseason score to 24-hour score for the head-injured players is not significant, whereas the comparable change for the student controls is statistically significant. The student controls also showed statistically significant improvement between 24 hours and 5 days, but showed no significant differences in the 5- to 10-day interval and the 10-day to post-season interval. On the other hand, the head-injured players demonstrated statistically significant improvement between 24 hours and 5 days and between 5 and 10 days, with no significant improvement in the 10-day to postseason interval.

To summarize these changes, we can say that the student controls displayed the normal testing behavior (i.e., there was an initial practice effect) which we would expect in serial administration of the PASAT 4 tests, whereas the head-injured players maintained a preseason baseline score (on average) in the preseason to 24-hour postinjury period, and then displayed an apparent recovery in the 24-hour to 5-day interval. These players continued to recover in the 5- to 10-day interval, and then leveled off throughout the remainder of the season to the post season. One sees the same pattern in Symbol Digit and PASAT 3 test performance but not in the performance on Trail Making tests A and B from the Halstead–Reitan battery (Tables 17–8 and 17–9). We are not certain at present why we do not see the same pattern on the Trail Making Test, but one possible explanation is that the practice effect for this test is so strong as to dilute the consequences of the injury on test performance. This hypothesis is being explored currently in further analyses.

Table 17–10 presents the percentage of head-injured players, players with orthopedic injuries, and student controls who reported selected symptoms at each assessment time. To summarize the table, we can state that there was a considerable increase in symptom reporting 24 hours post injury, compared to preseason symptom-reporting rates, for the head-injured players, which then diminished over time to return to the preseason rate by 10 days post injury. We do not observe the same pattern of symptom reporting for either the orthopedic group or the student controls. This gives us rather considerable evidence that the sequelae of mild injury are unique to head injury and not a consequence of general trauma or of population reporting rates for individuals

Table 17–10 Percent Reporting Selected Symptoms at Each Assessment

Symptom	Group	At Pre-Sn	At 24 hours	At 5 days	At 10 days	At Post-Sn
Headache	Head-injured	27	72	55	27	17
	Orthopedic	37	21	14	27	15
	Controls	27	11	17	10	15
Memory	Head-injured	03	35	27	08	03
	Orthopedic	04	04	02	00	07
	Controls	02	02	02	02	04
Nausea	Head-injured	03	31	24	08	03
	Orthopedic	06	02	05	11	11
	Controls	08	07	10	06	07
Dizziness	Head-injured	03	38	23	10	02
	Orthopedic	06	04	02	07	07
	Controls	04	09	06	02	00
Weakness	Head-injured	13	28	20	08	05
	Orthopedic	06	10	07	09	02
	Controls	10	09	12	12	07

of similar age and sex. We are currently performing formal statistical tests to support these interpretations.

In a broad sense, our study suggests that single mild head injury in football players often causes cognitive/information processing deficits which can be documented on neuropsychological assessment within 24 hours of the insult, and that rapid, although perhaps incomplete, recovery may take place over the next 5–10 days. These findings demonstrate similarities to general population mild head injury studies by Levin et al. (1987b) and McLean et al. (1984), who reported mild neuropsychological impairment within days and weeks of mild head trauma with rather rapid recovery of function. Earlier studies by Barth et al. (1983) and Rimel et al. (1981), in contrast, suggest that cognitive and psychosocial problems following such injuries may persist beyond three months post injury in clinical populations. As Dacey and Dikmen (1987) explain, the differences between these studies in severity of deficit and speed of recovery may be due to a variety of sample selection factors such as previous head injury, multiple trauma, alcohol use, psychosocial problems, and low premorbid intellectual/cognitive functioning. The Levin et al. (1987b) and McLean et al. (1984) studies eliminated or controlled for such factors, and for that reason evaluated much "cleaner" patients who are similar to this population of intelligent (estimated mean IQs of well over 100) and healthy, young athletes. Unfortunately, the football study, as well as other similar investigations, begins to break down in generalizability to the overall clinical mild head injury population since such trauma in the latter is more likely to be sustained by individuals who may have had previous cerebral insult and dysfunctional psychosocial histories (Barth et al., 1983; Rimel et al., 1981).

Another difficulty in generalizing from these football findings to other mild head trauma is that most (over 90%) of the reported football injuries reflected very mild trauma or "dings." The major mechanism of mild head injury in the general population is acceleration/deceleration in automobile accidents, which often results in more severe cerebral insult due to rotation and shear-strain.

What can be gleaned from this football study relates first and rather specifically to football itself:

1. Approximately 10% of all college football players will experience mild head injuries over any given season.
2. Over 40% of these athletes will have at least one mild head injury in their high school and college careers.
3. The effects of mild head injury may be measured by neuropsychological assessment.
4. Significant neuropsychological recovery will often take place in this healthy, young population within 10 days of their injury.

Questions still remain regarding the full extent of recovery and compensation, the short- and long-term effects of multiple head trauma, and factors predisposing a player to the risk of mild head injury.

Through further data review and analysis, it is our hope that we can provide the football community, and sports medicine psychologists in particular, with a brief and easily administered set of neuropsychological assessment tools that will aid team physicians in determining level of preseason information-processing skills in individual football players so that recovery of function following mild head injury can be closely monitored (along with other factors such as type of injury, history of previous injury, and reported postconcussion symptoms) to best determine risk for return to competition. Until our data are completely analyzed and further research is completed, a conservative approach to the practical question of when a player may return to competition is suggested, which emphasizes individual evaluation of the head injury circumstances including severity of trauma, number of previous head injuries, length of time since last cerebral insult, premorbid and present neuropsychological functioning, and recovery from postconcussion symptoms. Studying other football teams and sports with differing premorbid characteristics and mechanisms of injury, as well as appropriate control groups, will undoubtedly contribute to our better understanding of what we have now come to view as the spectrum of mild head trauma.

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